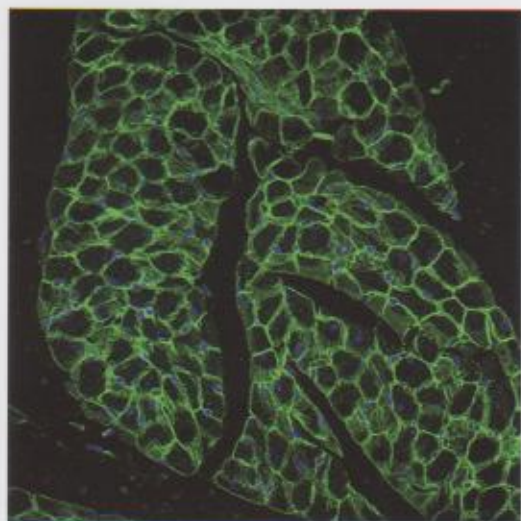


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**Diet
and the heart**

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Nutrient signaling and cardiovascular aging

Anna Planavila¹; Francesc Villarroya¹; Manlio Vinciguerra^{2,3}

¹Departament de Bioquímica i Biologia Molecular, Institut de Biomedicina (IBUB), Universitat de Barcelona and CIBER Fisiopatología de la Obesidad y Nutrición, Barcelona, Spain;

²Division of Medicine, University College London (UCL), UK;

³IRCCS “Casa Sollievo della Sofferenza” Hospital, San Giovanni Rotondo, Italy.

Correspondence: Manlio Vinciguerra, PhD, Senior Lecturer, University College London (UCL) – Division of Medicine, UCL Medical School - Royal Free Campus, 3rd Floor, Rowland Hill Street, London NW3 2PF, United Kingdom. E-mail: m.vinciguerra@ucl.ac.uk

Abstract

Aging is a major factor contributing to changes observed in the cardiovascular system in the elderly such as stiffening of the arterial tree and left ventricular diastolic function. These age-related changes in cardiovascular function lower the threshold at which cardiac diseases manifest. Evolutionarily conserved signals triggered by growth and hormones, eg, insulin and insulin-like growth factors, in response to the intake of specific macronutrients (proteins, carbohydrates, or fats), accelerate aging and mortality in animal models and humans. Dietary restriction, a ~25% reduction in calorie intake while maintaining micronutrients, by reducing the levels of aging-associated growth factors and hormones protects against cardiovascular diseases and against the decline in autonomic function. However, the role of nutrients and nutrient-associated signaling pathways in the decline in cardiac function and in the increase in cardiovascular diseases during aging is not completely understood. Here, we review the links between caloric intake, growth/hormonal factors, and intracellular signaling pathways in determining cardiac muscle (dys)function and regeneration during aging. ■ Heart Metab. 2014;63:4–7

Keywords: Aging; cardiac hypertrophy; cardiomyocytes; cardiomyopathy; cell signaling; growth factors; heart; laminin; mice models; nutrients.

The performance of the heart decreases with age, and this decrement in performance is a major risk factor for cardiovascular disease and mortality in the human population. However, the molecular pathways underlying cardiac aging are just beginning to be understood. In fact, aging results in a progressive functional and structural decline in the heart and arterial system. Age-dependent cardiac and vascular changes include cardiomyopathy,¹ impaired endothelial function and proliferation, increased stiffness of the arteries,^{2–4} left ventricular diastolic dysfunction, concentric hypertrophy, decreased systolic reverse,^{5,6} and diminished heart rate variability.⁷ Moreover, as a consequence of

the aging process, the interaction between the heart and arterial system adapts to preserve ventricle–arterial homeostasis. Hence, the age-associated structural and functional deterioration due to the intrinsic effects of aging on the myocardium together with the compensatory reactive cardiac modifications in response to the progressive increase of systolic load induced by elevated arterial stiffness, can have a detrimental effect on the aged heart. The view of the heart as a terminally differentiated postmitotic organ (Figure 1) that is unable to replace its damaged cells is shared by the scientific community and goes back to the 1960s. Recently, a dynamic concept of the heart in which cell